HHS Public Access

Author manuscript

J Nutr. Author manuscript; available in PMC 2016 April 06.

Published in final edited form as:

J Nutr. 2013 June ; 143(6): 995S–1000S. doi:10.3945/jn.112.173013.

Among ten sociodemographic and lifestyle variables, smoking is strongly associated with biomarkers of acrylamide exposure in a representative sample of the US population[1,,2,,3]

Hubert W. Vesper, **Maya R. Sternberg**, **Tunde Frame**, and **Christine M. Pfeiffer***

National Center for Environmental Health, CDC, Atlanta, GA

Abstract

Hemoglobin adducts of acrylamide (HbAA) and glycidamide (HbGA) have been measured as biomarkers of acrylamide exposure and metabolism in a nationally representative sample of the US population in the NHANES 2003–2004. We assessed the association of sociodemographic (age, sex, race-ethnicity, education, and income) and lifestyle variables (smoking, alcohol consumption, BMI, physical activity, and dietary supplement use) with these biomarkers in US adults (≥20 y). We used bivariate and multiple regression models and assessed the magnitude of an estimated change in biomarker level with change in a covariable for 2 biomarkers of acrylamide exposure.

Smoking was strongly and significantly correlated with HbAA and HbGA levels (r_S=0.51 and 0.42, respectively), with biomarker concentrations being 126% and 101% higher in smokers compared to nonsmokers after adjusting for sociodemographic and lifestyle covariates. Age was moderately and significantly correlated with both biomarkers (r_S=−0.21 and −0.22, respectively). BMI (r_S=−0.11) and alcohol consumption (r_S=0.13) were weakly yet significantly correlated with HbAA levels only. The estimated percent change in biomarker concentration was ≤20% for all variables other than smoking after adjusting for sociodemographic and lifestyle covariates. Using multiple regression models, the sociodemographic variables explained 9% and 7%, while the sociodemographic and lifestyle variables together explained 46% and 25% of the variability in HbAA and HbGA, respectively, showing the importance of considering and adequately controlling for these variables in future studies. Our findings will be useful in the design and analysis of future studies that assess and evaluate exposure to acrylamide and its metabolism to glycidamide.

---

[1]No specific sources of financial support. The findings and conclusions in this report are those of the authors and do not necessarily represent the official views or positions of the Centers for Disease Control and Prevention/Agency for Toxic Substances and Disease Registry or the Department of Health and Human Services.

[2]Author disclosures: HW Vesper, MR Sternberg, T Frame, CM Pfeiffer, no conflicts of interest.

[3]Supplemental Tables 1–2 are available from the “Online Supporting Material” link in the online posting of the article and from the same link in the online table of contents at http://jn.nutrition.org.

*To whom correspondence should be addressed: Christine M. Pfeiffer, Division of Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention, 4770 Buford Hwy, NE, Mail Stop F55, Atlanta, GA 30341, CPfeiffer@cdc.gov.

Keywords

NHANES; acrylamide; glycidamide; hemoglobin adducts; education; poverty income ratio; smoking; alcohol consumption; BMI; physical activity; supplement use

INTRODUCTION

Acrylamide occurs in a wide range of food products that are commonly consumed by a large portion of the population. It is formed in food during frying or baking from the reaction of reducing sugars such as glucose with the amino acid asparagine via the Maillard reaction (1, 2, 3). Among different food products, potato chips, French fries and some baked goods were found to contain high amounts of acrylamide (4, 5). Dietary intake is considered one major source of acrylamide exposure in the general population. Exposure to acrylamide is of concern, because acrylamide is a suspected human carcinogen and a potentially endocrine disrupting chemical (6, 7, 8, 9). These concerns initiated a wide range of research to assess intake, possible health risks, and to minimize acrylamide content in foods (10, 11, 12, 13).

Acrylamide is metabolized in the liver to its epoxide, glycidamide, which is primarily mediated by cytochrome P450 2E1 (14). Glycidamide forms DNA adducts and thus is considered genotoxic (15, 16, 17). Therefore, knowledge about exposure to acrylamide and its metabolite is important to assess and interpret potential health effects.

Adducts of acrylamide (HbAA)[4] and glycidamide (HbGA) with hemoglobin, so called hemoglobin adducts, have been successfully used as biomarkers to assess acrylamide exposure in humans (18, 19, 20). These adducts reflect the time weighted exposure of acrylamide in a person over the past 4 mo.

CDC's *Second National Report on Biochemical Indicators of Diet and Nutrition in the US Population* provides a descriptive analysis of the HbAA and HbGA levels of Americans by age, sex, and race-ethnicity for data from the NHANES 2003–2004. These analyses however, cannot explain why there are differences in these exposure biomarker levels among demographic subgroups (21).

Dietary intake and smoking are generally understood to be the 2 important determinants of HbAA and HbGA biomarker concentrations in blood. Several studies have investigated the relationship between dietary and smoking exposure and these biomarkers (20, 22, 24, 25, 26, 27, 28). However, to our knowledge, no studies have systematically examined the association of a panel of sociodemographic and lifestyle variables on HbAA and HbGA levels in the US population. The objective of our analysis was to assess the association of 10 selected sociodemographic and lifestyle variables with these biomarkers.

[4]Abbreviations used: HbAA, hemoglobin adduct of acrylamide; HbGA hemoglobin adduct of glycidamide; NCHS, National Center for Health Statistics; PIR, poverty income ratio.

SUBJECTS AND METHODS

Survey design and participants

The NHANES collects cross-sectional data on the health and nutrition status of the civilian non-institutionalized US population (29). Participants in NHANES 2003–2004, aged ≥20 y (n = 4152) who had a stored blood specimen available for analysis of HbAA and HbGA constituted the study sample. All respondents gave their informed consent, and the NHANES protocol was reviewed and approved by the NCHS Research Ethics Review Board.

Laboratory methods

HbAA and HbGA were measured in EDTA whole blood as described previously (27, 30). The detection limits for HbAA and HbGA adducts were 3 and 4 pmol/g of Hb, respectively. The inter-day imprecision (n = 20 d) of this method, expressed as percent CV, was on average 13% for HbAA and 19% for HbGA, determined with 3 blood pools.

Sociodemographic and lifestyle variables

For bivariate analyses, we categorized the variables as follows: age (20–39 y, 40–59 y, and ≥60 y); race-ethnicity (non-Hispanic white [NHW], non-Hispanic black [NHB], and Mexican American [MA]); education (<high school, high school, and >high school); family poverty income ratio (PIR; 0–1.85 [low], >1.85–3.5 [medium], and >3.5 [high], (31)); smoking status (serum cotinine ≤10 μg/L [nonsmoker], >10 μg/L [smoker], (32)); alcohol consumption (average daily number of “standard” drinks [1 drink ≈ 15 g ethanol]; no drinks, <1 drink (not 0), 1-<2 drinks, ≥2 drinks); BMI (kg/m^2) (<18.5 [underweight], ≥18.5 and <25.0 [normal], ≥25.0 and <30.0 [overweight], ≥30.0 [obese], (33)); physical activity (calculated as total metabolic equivalent task (MET)-min/wk from self-reported leisure time physical activities; no leisure time physical activities, 0–<500, 500–<1000, ≥1000 MET-min/wk); supplement use (reported taking a dietary supplement within the past 30 d; yes [user], no [non-user]).

Statistical analyses

As we used the same statistical methods for the series of papers presented in this supplement, the reader is referred to Sternberg *et al.* (34) for a detailed description of the methods and for a discussion of compromises taken in developing the multiple regression model due to the limited degrees of freedom, such as the number of covariates considered, the chosen form of continuous covariates, and the consideration of interactions between covariates. Bivariate associations for categorical variables were assessed by calculating the geometric means and 95% CI for each category and Spearman correlations for selected continuous variables. Linear regression analyses were used to assess the confounding effects and to determine whether statistical significance persisted after adjusting for differences in key variables. HbAA and HbGA data were log transformed based on the distribution of the biomarker. The covariates were arranged into 2 chunks: sociodemographic variables (age, sex, race-ethnicity, education level, and PIR) and lifestyle variables (smoking, alcohol consumption, BMI, physical activity, and dietary supplement use) and entered hierarchically.

We summarized the results of each model and showed the magnitude of association by presenting the percent change in biomarker concentrations with change in each covariate holding all other remaining covariates constant for each model. All estimates were weighted to account for the unequal probabilities of selection and adjustment for non-response. Two-sided *P*-values were flagged as statistically significant if $P < 0.05$.

RESULTS

HbAA and HbGA data were available for 4093 and 4152 NHANES participants, respectively. The study population was estimated to consist of 48% men and 71% nonsmokers. The proportion of NHW, NHB and MA was 72%, 11% and 8%, respectively (Table 1).

Spearman correlation coefficients showed that smoking was strongly and significantly correlated with HbAA ($r_s = 0.51$) and HbGA ($r_s = 0.42$), while other variables showed no or only weak correlations ($|r_s| \leq 0.2$, $P < 0.05$) (Table 2). Using bivariate methods (model 1), all sociodemographic variables were significantly but weakly associated with HbAA and all but sex and PIR were significantly but weakly associated with HbGA (Table 3). Values for both biomarkers were lower among persons with higher age, educational level and PIR. NHB had higher HbAA and lower HbGA values compared to the other 2 race-ethnic groups and men had higher HbAA values compared to women. Most lifestyle variables except for smoking were either weakly or not associated with biomarkers of acrylamide exposure (Table 4). Smoking was associated with at least 100% higher hemoglobin adduct values compared to not smoking; supplement use with ~20% higher values for both biomarkers; and increasing consumption of alcohol with higher levels of HbAA. Physical activity was not associated with either marker of acrylamide exposure, while higher BMI was associated with lower levels of HbAA only.

Using multiple regression models, the sociodemographic variables (model 2) explained 9% of the variability in biomarker values for HbAA and 7% for HbGA (Supplemental Table 1). Together, the sociodemographic and lifestyle variables (model 3) explained 46% and 25% of the HbAA and HbGA variability, respectively. In all models, age, smoking, supplement use, and BMI remained significantly associated with HbAA levels after adjusting for sociodemographic and lifestyle variables (Supplemental Table 1). Age, smoking, and race (NHB vs. NHW only) remained significantly associated with HbGA levels. Sex and alcohol consumption were significantly associated with HbGA after controlling for both sociodemographic and lifestyle variables.

Because the log transformations may obscure the interpretation of the *beta* coefficients, we estimated the percent change in biomarker levels associated with each covariable (Fig. 1 and Supplemental Table 2). Smoking had a strong association with both biomarkers, with estimated biomarker levels in smokers being ~100% higher for HbGA and ~130% higher for HbAA compared to nonsmokers after adjusting for sociodemographic and lifestyle variables. All other estimated percent changes were comparatively moderate (≤20%) and generally not consistent between HbAA and HbGA, except for age which was associated

with slightly but significantly lower HbAA (3%) and HbGA (6%) values with every 10 y increase (model 3).

DISCUSSION

Among the 10 sociodemographic and lifestyle variables investigated in this representative sample of US adults, smoking showed a strong association with both biomarkers of acrylamide exposures and remained a significant variable in all regression models. Smokers had at least twice the HbAA and HbGA levels compared to nonsmokers which is consistent with other studies (27, 35). Among the sociodemographic variables, we found age to be significantly negatively associated with HbAA and HbGA levels in all models, suggesting that acrylamide exposure as well as metabolism may change with age.

In our full regression model, race (NHB vs. NHW only) and sex were significant correlates for HbGA but not for HbAA, suggesting differences in acrylamide metabolism rather than in acrylamide exposure. Different polymorphisms for CYP 2E1 and their impact on acrylamide metabolism have been described (36, 37, 38). However, no information is available about the occurrence and frequency of these polymorphisms in this study population. Similarly, sex differences in the pharmacologic activity of CYP 2E1 seem to exist (39, 40), but the impact of these differences on acrylamide is not fully understood.

Our analysis found that BMI was significantly negatively associated with HbAA levels, which is consistent with a previous study (35). The reason for the negative association is not fully understood and requires further investigation.

Alcohol consumption was significantly negatively associated with HbGA values in our analysis only after adjusting for sociodemographic and lifestyle variables, which appears consistent with observations in other study populations (30, 41). Alcohol induces CYP 2E1, which metabolizes alcohol as well as acrylamide (42). The negative association could be explained with a competitive effect of alcohol and acrylamide as substrate.

While sociodemographic variables alone explained only 9% of the variability in HbAA and 7% for HbGA, the full regression model 3 which also included lifestyle variables explained 46% of HbAA and 25% of HbGA variability. These findings seem consistent with a previous study that investigated the association of food intake on biomarker levels using data from the same NHANES survey, but included children as well (43).

To our knowledge, this is the first study that examined the association and cumulative effects of demographic, socioeconomic, and lifestyle variables on HbAA and HbGA levels. By applying a systematic modeling approach, we were able to assess the magnitude of an estimated change in biomarker level with change in covariable across biomarkers. Furthermore, the findings for the acrylamide biomarkers can also be compared to findings for other classes of diet and nutrition biomarkers reported in accompanying papers in this supplement. The use of a chunk test in our modeling approach maintains the interpretation of the *beta* coefficients and *P*-values and therefore is less likely to yield false positive findings that are associated with automatic variable selection methods like backwards elimination.

The limitations in our study are the lack of testing for interactions between individual variables, which was not performed due to limitations in degrees of freedom. We did not study biological variables or how dietary intake is associated with HbAA and HbGA or interacts with variables included in our analysis. However, dietary acrylamide intake estimates were found to correlate only weakly with HbAA and HbGA (20, 22, 25). This could be explained with the high variability in acrylamide content within food groups, the different exposure periods covered by food intake questionnaires and biomarkers, and incomplete acrylamide data in foods. Our analysis was designed to examine associations of HbAA and HbGA with selected variables after adjusting for sociodemographic and lifestyle variables. In this context, acrylamide intake would be considered an outcome variable as opposed to a covariate.

In summary, we conclude that smoking, age and BMI were the 3 variables that were related with HbAA and that smoking, race and alcohol consumption were important correlates for HbGA. The results from this descriptive modeling analysis will help future data analyses that set out to build predictive models to answer specific research questions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The authors acknowledge technical assistance from Bridgette Haynes and Yi Pan and contributions from the following laboratory members: Christina Waters, Judith Heitz, Antoinette Smith, Ashley Ribera, Marcela Muresan, Ashley Tippins, Cindy Tse, and Monir Clark. C.M.P and M.R.S. designed the overall research project with input from H.W.V.; H.W.V. and M.R.S. conducted most of the research; M.R.S. analyzed most of the data; and H.W.V. wrote the initial draft, which was modified after feedback from all coauthors, and had primary responsibility for content. All authors read and approved the final manuscript.

References

1. Pedreschi F, Kaack K, Granby K. Reduction of acrylamide formation in potato slices during frying. LWT - Food Sci Technol. 2004; 37(6):679–85.
2. Mottram DS, Wedzicha BL, Dodson AT. Acrylamide is formed in the Maillard reaction. Nature. 2002; 419:448–9. [PubMed: 12368844]
3. Stadler RH, Blank I, Varga N, Robert F, Hau J, Guy PA, Robert MC, Riediker S. Acrylamide from Maillard reaction products. Nature. 2002; 419:449–50. [PubMed: 12368845]
4. Tareke E, Rydberg P, Karlsson P, Eriksson S, Törnqvist M. Analysis of acrylamide, a carcinogen formed in heated foodstuffs. J Agric Food Chem. 2002; 50:4998–5006. [PubMed: 12166997]
5. FDA. Survey Data on Acrylamide in Food: Total Diet Study Results. [cited 2012 Sept 6]. Available from: http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/ChemicalContaminants/Acrylamide/ucm053566.htm#table4
6. NTP. Report on Carcinogens. Twelfth. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program; 2011. p. 25-8.
7. IARC (International Agency for Research on Cancer). Acrylamide. IARC Monogr Eval Carcinog Risk Hum. 1995; 60:1–45.
8. Hamdy SM, Bakeer HM, Eskander EF, Sayed ON. Effect of acrylamide on some hormones and endocrine tissues in male rats. Hum Exp Toxicol. 2012; 31:483–91. [PubMed: 21878450]
9. Camacho L, Latendresse JR, Muskhelishvili L, Patton R, Bowyer JF, Thomas M, Doerge DR. Effects of acrylamide exposure on serum hormones, gene expression, cell proliferation, and

histopathology in male reproductive tissues of Fischer 344 rats. Toxicol Lett. 2012; 211:135–43. [PubMed: 22459607]

10. Lineback DR, Coughlin JR, Stadler RH. Acrylamide in foods: a review of the science and future considerations. Annu Rev Food Sci Technol. 2012; 3:15–35. [PubMed: 22136129]
11. National Toxicology Program. Report on Carcinogens. 12th2011. [cited 2012 Sept 6]. Available from: http://www.niehs.nih.gov/news/sya/sya-roc/
12. Pruser KN, Flynn NE. Acrylamide in health and disease. Front Biosci (Schol Ed). 2011; 3:41–51. [PubMed: 21196355]
13. Pelucchi C, Galeone C, Talamini R, Negri E, Polesel J, Serraino D, La Vecchia C. Dietary acrylamide and pancreatic cancer risk in an Italian case–control study. Ann Oncol. 2011; 22:1910–5. [PubMed: 21285136]
14. Sumner SC, Fennell TR, Moore TA, Chanas B, Gonzalez F, Ghanayem BI. Role of cytochrome P450 2E1 in the metabolism of acrylamide and acrylonitrile in mice. Chem Res Toxicol. 1999; 12:1110–6. [PubMed: 10563837]
15. Watzek N, Böhm N, Feld J, Scherbl D, Berger F, Merz KH, Lampen A, Reemtsma T, Tannenbaum SR, Skipper PL, Baum M, Richling E, Eisenbrand G. N7-glycidamide-guanine DNA adduct formation by orally ingested acrylamide in rats: a dose-response study encompassing human diet-related exposure levels. Chem Res Toxicol. 2012; 25:381–90. [PubMed: 22211389]
16. Gamboa da Costa G, Churchwell MI, Hamilton LP, Von Tungeln LS, Beland FA, Marques MM, Doerge DR. DNA adduct formation from acrylamide via conversion to glycidamide in adult and neonatal mice. Chem Res Toxicol. 2003; 16:1328–37. [PubMed: 14565774]
17. Doerge DR, Young JF, McDaniel LP, Twaddle NC, Churchwell MI. Toxicokinetics of acrylamide and glycidamide in Fischer 344 rats. Toxicol Appl Pharmacol. 2005; 208:199–209. [PubMed: 16239164]
18. Ogawa M, Oyama T, Isse T, Yamaguchi T, Murakami T, Endo Y, Kawamoto T. Hemoglobin adducts as a marker of exposure to chemical substances, especially PRTR class I designated chemical substances. J Occup Health. 2006; 48(5):314–28. [PubMed: 17053297]
19. Törnqvist M, Fred C, Haglund J, Helleberg H, Paulsson B, Rydberg P. Protein adducts: quantitative and qualitative aspects of their formation, analysis and applications. J Chromatogr B. 2002; 778(1–2):279–308.
20. Wilson KM, Vesper HW, Tocco P, Sampson L, Rosén J, Hellenäs KE, Törnqvist M, Willett WC. Validation of a food frequency questionnaire measurement of dietary acrylamide intake using hemoglobin adducts of acrylamide and glycidamide. Cancer Causes Control. 2008; 20:269–78. [PubMed: 18855107]
21. Pfeiffer CM, Sternberg MR, Schleicher RL, Haynes BMH, Rybak ME, Pirkle JL. CDC's Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population is a valuable tool for researchers and policy makers to assess nutritional status. J Nutr. Submitted for review as part of supplement.
22. Bjellaas T, Olesen PT, Frandsen H, Haugen M, Stølen LH, Paulsen JE, Alexander J, Lundanes E, Becher G. Comparison of estimated dietary intake of acrylamide with hemoglobin adducts of acrylamide and glycidamide. Toxicol Sci. 2007; 98:110–7. [PubMed: 17449897]
23. Kutting B, Uter W, Drexler H. The association between self-reported acrylamide intake and hemoglobin adducts as biomarkers of exposure. Cancer Causes Control. 2008; 19:273–81. [PubMed: 17985202]
24. Olesen PT, Olsen A, Frandsen H, Frederiksen K, Overvad K, Tjønneland A. Acrylamide exposure and incidence of breast cancer among postmenopausal women in the Danish Diet, Cancer and Health Study. Int J Cancer. 2008; 122:2094–100. [PubMed: 18183576]
25. Wirfalt E, Paulsson B, Tornqvist M, Axmon A, Hagmar L. Associations between estimated acrylamide intakes, and hemoglobin AA adducts in a sample from the Malmo Diet and Cancer cohort. Eur J Clin Nutr. 2008; 62:314–23. [PubMed: 17356560]
26. Hagmar L, Wirfalt E, Paulsson B, Törnqvist M. Differences in hemoglobin adduct levels of acrylamide in the general population with respect to dietary intake, smoking habits and gender. Mutat Res. 2005; 580:157–65. [PubMed: 15668117]

27. Vesper HW, Bernert JT, Ospina M, Meyers T, Ingham L, Smith A, Myers GL. Assessment of the relation between biomarkers for smoking and biomarkers for acrylamide exposure in humans. Cancer Epidemiol Biomarkers Prev. 2007; 16:2471–8. [PubMed: 18006939]
28. Scherer G, Engl J, Urban M, Gilch G, Janket D, Riedel K. Relationship between machine-derived smoke yields and biomarkers in cigarette smokers in Germany. Regul Toxicol Pharmacol. 2007; 47:171–83. [PubMed: 17034917]
29. U.S. Centers for Disease Control and Prevention. About the National Health and Nutrition Examination Survey. [cited 2012 Aug 24]. Available from: http://www.cdc.gov/nchs/nhanes.htm
30. Vesper HW, Slimani N, Hallmans G, Tjonneland A, Agudo A, Benetou V, Bingham S, Boeing H, Boutron-Ruault MC, et al. Cross-sectional study on acrylamide hemoglobin adducts in subpopulations from the European Prospective Investigation into Cancer and Nutrition (EPIC) Study. J Agric Food Chem. 2008; 56:6046–53. [PubMed: 18624432]
31. U.S. Centers for Disease Control and Prevention. NHANES analytic guidelines, the Third National Health and Nutrition Examination Survey, NHANES III 1988–94. Hyattsville (MD): National Center for Health Statistics; Oct. 1996 [cited 2012 Sept 11]. Available from: http://www.cdc.gov/nchs/data/nhanes/nhanes3/nh3gui.pdf
32. Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the US population to environmental tobacco smoke. J Am Med Assoc. 1996; 275:1233–40.
33. WHO. Report of a WHO Consultation. Geneva: World Health Organization; 2000. Obesity: preventing and managing the global epidemic. WHO Technical Report Series 894
34. Sternberg MR, Schleicher RL, Pfeiffer CM. Regression modeling plan for twenty-nine biochemical indicators of diet and nutrition measured in NHANES 2003–2006. J Nutr. Submitted for review as part of supplement.
35. Vesper HW, Caudill SP, Osterloh JD, Meyers T, Scott D, Myers GL. Exposure of the U.S. population to acrylamide in the National Health and Nutrition Examination Survey 2003–2004. Environ Health Perspect. 2010; 118:278–83. [PubMed: 20123601]
36. Duale N, Bjellaas T, Alexander J, Becher G, Haugen M, Paulsen JE, Frandsen H, Olesen PT, Brunborg G. Biomarkers of human exposure to acrylamide and relation to polymorphisms in metabolizing genes. Toxicol Sci. 2009; 108:90–9. [PubMed: 19131562]
37. Huang YF, Chen ML, Liou SH, Chen MF, Uang SN, Wu KY. Association of CYP2E1, GST and mEH genetic polymorphisms with urinary acrylamide metabolites in workers exposed to acrylamide. Toxicol Lett. 2011; 203:118–26. [PubMed: 21402133]
38. Krishnakumar D, Gurusamy U, Dhandapani K, Surendiran A, Baghel R, Kukreti R, Gangadhar R, Prayaga U, Manjunath S, Adithan C. Genetic polymorphisms of drug-metabolizing phase I enzymes CYP2E1, CYP2A6 and CYP3A5 in South Indian population. Fundam Clin Pharmacol. 2012; 26:295–306. [PubMed: 21265876]
39. Anderson GD. Gender differences in pharmacological response. Int Rev Neurobiol. 2008; 83:1–10. [PubMed: 18929073]
40. Scandlyn MJ, Stuart EC, Rosengren RJ. Sex-specific differences in CYP450 isoforms in humans. Expert Opin Drug Metab Toxicol. 2008; 4:413–24. [PubMed: 18524030]
41. Vikström AC, Wilson KM, Paulsson B, Athanassiadis I, Grönberg H, Adami HO, Adolfsson J, Mucci LA, Bälter K, Törnqvist M. Alcohol influence on acrylamide to glycidamide metabolism assessed with hemoglobin-adducts and questionnaire data. Food Chem Toxicol. 2010; 48:820–4. [PubMed: 20034532]
42. Oneta CM, Lieber CS, Li J, Rüttimann S, Schmid B, Lattmann J, Rosman AS, Seitz HK. Dynamics of cytochrome P4502E1 activity in man: induction by ethanol and disappearance during withdrawal phase. J Hepatol. 2002; 36:47–52. [PubMed: 11804663]
43. Tran NL, Barraj LM, Murphy MM, Bi X. Dietary acrylamide exposure and hemoglobin adducts–National Health and Nutrition Examination Survey (2003–04). Food Chem Toxicol. 2010; 48:3098–108. [PubMed: 20696196]

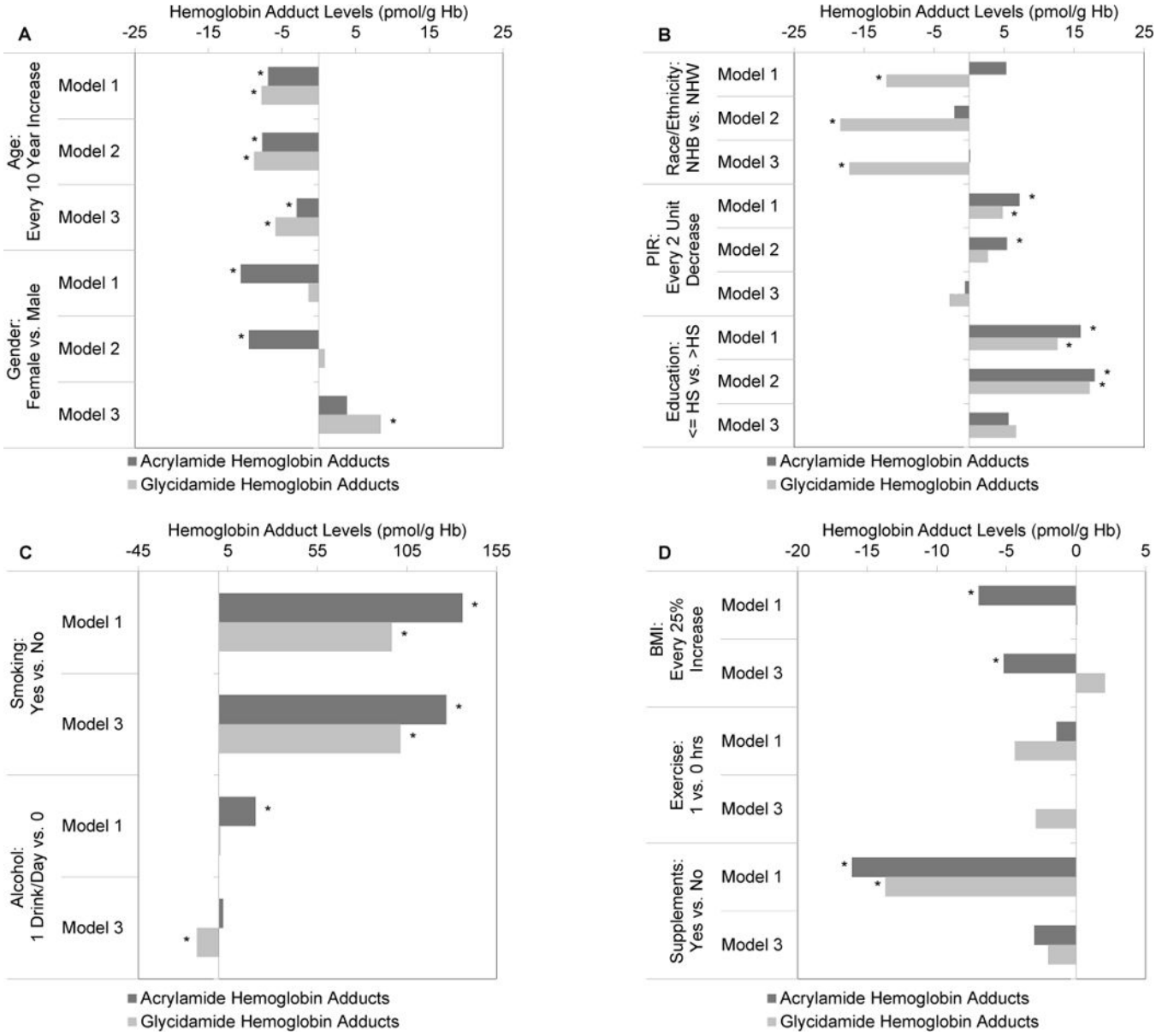

Figure 1.

Estimated change in hemoglobin adduct of acrylamide and glycidamide with change in sociodemographic and lifestyle variables for adults ≥20 y, NHANES 2003–2004

Model 1 represents simple linear regression, model 2 represents multiple linear regression with sociodemographic factors, and model 3 represents multiple linear regression with sociodemographic and lifestyle factors; changes derived from a linear regression model while holding any other variables in the model constant; asterisk accompanying the percent change indicates significance (P <0.05).

NHB, non-Hispanic black; NHW, non-Hispanic white; HS, high school; PIR, poverty income ratio.

Table 1

Descriptive information for the adult US population aged ≥20 y by sociodemographic and lifestyle variables, NHANES 2003–2004

Variable	Category	Estimate[1]	Sample size[2]	
			Acrylamide hemoglobin adduct	Glycidamide hemoglobin adduct
Age, y	20–39	38.8	1406	1446
	40–59	38.5	1164	1177
	≥60	22.7	1523	1529
Sex	Male	48.0	1974	2008
	Female	52.1	2119	2144
Race-ethnicity	Mexican American	7.76	862	867
	Non-Hispanic black	11.2	748	787
	Non-Hispanic white	72.1	2182	2203
Education	<High school	30	1213	1229
	High school	25	1039	1047
	>High school	45	1834	1870
PIR[3]	Low (0–1.85)	31.4	1664	1702
	Middle (>1.85–3.5)	27.6	1012	1016
	High (>3.5)	41.1	1192	1207
Supplement use[4]	No	70.2	2106	2183
	Yes	29.8	1924	1960
Smoking status[5]	No	71.2	2987	3013
	Yes	28.9	1067	1098
Alcohol consumption[6]	No drinks	30.6	1413	1596
	<1 (not 0)	56.6	1954	909
	1–<2	7.6	237	493
	≥2	5.2	184	989
BMI[7]	Underweight	1.73	57	56
	Normal weight	32.0	1217	1225
	Overweight	34.1	1431	1438
	Obese	32.2	1303	1347
Physical activity[8]	None reported	33.0	1588	1596
	0–<500	25.0	894	909
	500–<1000	13.6	483	493
	≥1000	28.4	964	989

[1] Estimates provided are percent (%)

[2] Values represent unweighted sample sizes

[3] PIR, family poverty income ratio; low: 0–1.85; medium: >1.85–3.5; high: >3.5

[4] "Supplement user" defined as participant who reported taking a dietary supplement within the past 30 d

[6] Alcohol consumption: calculated as average daily number of "standard" drinks [(quantity × frequency)/365.25]; 1 drink ≈ 15 g ethanol

[7] BMI (kg/m^2) definitions: underweight: <18.5; normal weight: 18.5–<25; overweight: 25–<30; and obese: ≥30

[8] Physical activity: calculated as total metabolic equivalent task (MET)-min/wk from self-reported leisure time physical activities

Table 2

Spearman correlation coefficients describing bivariate associations between hemoglobin adducts of acrylamide and glycidamide and selected continuous sociodemographic and lifestyle variables for adults ≥20 y, NHANES 2003–2004

Variable	Acrylamide hemoglobin adduct	Glycidamide hemoglobin adduct
Age	−0.21 *	−0.22 *
PIR [1]	−0.06 *	−0.07 *
Smoking	0.51 *	0.42 *
Alcohol consumption	0.13 *	−0.03
BMI	−0.11 *	0.00
Physical activity	0.02	−0.04

* Significant correlation; $P < 0.05$

[1] PIR, family poverty income ratio

Table 3

Unadjusted biomarker levels by sociodemographic variable categories for adults ≥20 y, NHANES 2003–2004[1]

Variable		Acrylamide hemoglobin adduct *pmol/g Hb*	Glycidamide hemoglobin adduct *pmol/g Hb*
Age, *y*	20–39	68.5 (64.1 – 73.3)	65.0 (61.4 – 68.9)
	40–59	64.0 (59.9 – 68.4)	60.1 (56.8 – 63.5)
	≥60	50.1 (47.9 – 52.3)	45.5 (42.8 – 48.3)
P-value[2]		*<0.0001*	*<0.0001*
r^2 (%)[3]		*4*	*4*
Sex	Men	65.9 (61.5 – 70.5)	58.6 (55.7 – 61.7)
	Women	58.9 (55.7 – 62.2)	57.8 (54.4 – 61.4)
P-value		*<0.0001*	*0.54*
r^2 (%)		*1*	*< 1*
Race-ethnicity[4]	MA	62.7 (59.3 – 66.3)	62.9 (59.0 – 67.0)
	NHB	66.5 (58.0 – 76.2)	52.9 (49.4 – 56.7)
	NHW	63.1 (59.2 – 67.3)	60.0 (56.1 – 64.1)
P-value		*<0.0001*	*<0.0001*
r^2 (%)		*2*	*1*
Education	<High school	65.0 (58.9 – 71.6)	59.0 (54.4 – 63.9)
	High school	69.0 (62.7 – 75.9)	64.3 (59.6 – 69.4)
	>High school	58.1 (55.1 – 61.3)	55.2 (52.1 – 58.5)
P-value		*0.0004*	*0.0009*
r^2 (%)		*2*	*1*
PIR[5]	Low	66.7 (62.7 – 70.9)	60.3 (56.9 – 63.9)
	Medium	61.5 (57.7 – 65.6)	59.2 (56.8 – 61.8)
	High	59.2 (55.9 – 62.8)	56.4 (53.3 – 59.6)
P-value		*<0.0001*	*0.06*
r^2 (%)		*1*	*< 1*

[1] Values represent geometric means (95% CI)

[2] *P*-value based on Wald F test, which tests whether at least 1 of the means across the categories is significantly different

[3] r^2 based on model 1, simple linear regression, using categories as shown

[4] MA, Mexican American; NHB, non-Hispanic black; NHW, non-Hispanic white

[5] PIR, family poverty income ratio; low: 0–1.85; medium: >1.85–3.5; high: >3.5

Table 4

Unadjusted biomarker levels by lifestyle variable categories for adults ≥20 y, NHANES 2003–2004[1]

Variable		Acrylamide hemoglobin adduct *pmol/g Hb*	Glycidamide hemoglobin adduct *pmol/g Hb*
Supplement use[2]	No	57.4 (54.8 – 60.1)	54.5 (51.5 – 57.5)
	Yes	68.4 (63.3 – 73.9)	63.1 (59.9 – 66.4)
P-value[3]		*<0.0001*	*<0.0001*
r^2 (%)[4]		*2*	*1*
Smoking[5]	No	48.2 (46.4 – 50.1)	47.7 (45.5 – 49.9)
	Yes	114 (104 – 125)	93.7 (86.7 – 101)
P-value		*<0.0001*	*<0.0001*
r^2 (%)		*42*	*20*
Alcohol consumption[6]	No drinks	54.4 (50.7 – 58.4)	54.9 (51.4 – 58.7)
	<1 (not 0)	63.0 (59.4 – 66.7)	59.7 (56.5 – 63.0)
	1–<2	79.1 (71.4 – 87.6)	64.2 (56.2 – 73.4)
	≥2	84.4 (70.1 – 102)	55.8 (45.9 – 67.9)
P-value		*<0.0001*	*0.0245*
r^2 (%)		*4*	*4*
BMI[7]	Underweight	80.4 (58.5 – 110)	55.7 (39.2 – 79.0)
	Normal	67.4 (62.8 – 72.3)	59.1 (55.4 – 63.0)
	Overweight	60.9 (56.9 – 65.1)	57.3 (52.9 – 62.1)
	Obese	58.1 (54.5 – 61.9)	58.7 (55.7 – 61.7)
P-value		*0.0002*	*0.89*
r^2 (%)		*1*	*0*
Physical activity[8]	None reported	64.1 (59.2 – 69.5)	59.5 (55.2 – 64.2)
	0–<500	61.5 (58.2 – 64.9)	60.4 (56.6 – 64.4)
	500–<1000	58.8 (54.0 – 63.9)	55.2 (49.7 – 61.4)
	≥1000	61.8 (57.9 – 66.1)	56.5 (52.8 – 60.5)
P-value		*0.23*	*0.06*
r^2 (%)		*< 1*	*< 1*

[1] Values represent geometric means (95% CI)

[2] "Supplement user" defined as participant who reported taking any dietary supplement within the past 30 d

[3] *P*-value based on Wald F test, which tests whether at least one of the means across the categories is significantly different

[4] r^2 based on model 1, simple linear regression, using categories as shown

[5] "Smoker" defined by serum cotinine concentration >10 μg/L; ≤10 μg/L defined as "nonsmoker"

[6] Alcohol consumption (drinks/d): calculated as average daily number of "standard" drinks [(quantity × frequency)/365.25]; 1 drink ≈ 15 g ethanol

[7] BMI (kg/m^2) definitions: underweight: <18.5; normal weight: 18.5–<25; overweight: 25–<30; and obese: ≥30

[8] Physical activity: calculated as total metabolic equivalent task (MET)-min/wk from self-reported leisure time physical activities